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CASE REPORT - NEUROSURGERY GENERAL

Idiopathic chronic temporal lobe herniation with associated epilepsy

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Abstract
Herniation of parahippocampal gyrus is usually caused by pressure differentials intracranially, and herniation without known risk factors is extremely rare. We describe a patient with a long history of seizures and a remote status epilepticus event. On magnetic resonance imaging, a presumed left temporal lobe tumor was observed. On neurosurgical consultation, the lesion was identified as a chronic mesial temporal lobe herniation. The patient lacked history that would suggest risk of cerebral herniation. Accurately identifying the patient's chronic temporal lobe herniation radiographically likely saved this patient from unnecessary surgery or biopsy and allowed the patient to receive appropriate conservative care.

Keywords Temporal lobe · Herniation · Epilepsy · Seizure

Abbreviations
CSF Cerebrospinal fluid
MRI Magnetic resonance imaging
TLE Temporal lobe epilepsy

Introduction

Lesions of the cerebellopontine angle and the parahippocampal and hippocampal gyrus region are not uncommon and can include extra-axial lesions (such as schwannomas, meningiomas, and epidermoid and dermoid cysts) and intrinsic lesions (such as astrocytomas, gangliogliomas, and hamartomas) [3]. Symptoms often depend on lesion size, associated brain compression, and cranial nerve involvement. Lesions in this area warrant neurosurgical evaluation.

Brain herniation, the movement of brain parenchyma from one cranial compartment to another, is a serious and sometimes fatal condition that requires emergent action to counteract such disastrous brain processes [7]. The herniation is caused by an altered compartmental intracranial pressure stemming from cerebrospinal fluid (CSF) leak, trauma, infection, hydrocephalus, pneumocephalus, edema, intracranial hemorrhage, or brain tumor [6]. Cases of brain herniation without known acute or chronic intracranial compartment pressure differentials are rare [6].

Chronic herniation syndromes beyond tonsillar herniation are not well recognized [11]. Chronic transtentorial uncal herniation has been described in a few case reports, but none have been reported without prior risk factors such as CSF shunting, Dandy-Walker syndrome, traumatic brain injury, or other causes described above [2, 5, 11–13]. We describe a patient with a long-standing history of epilepsy with chronic transtentorial uncal herniation. The pathology was originally misdiagnosed as a tumor on the initial magnetic resonance imaging (MRI).

Case report

A 58-year-old, right-hand dominant, man with a complex seizure history presented with shuffling gait and tremor. On MRI at an outside hospital, he was found to have a $13.4 \times 35.1 \times 11.4$-mm lesion in the left posterior fossa (Fig. 1). This lesion was initially thought to be a parahippocampal tumor, and the patient was referred for neurosurgical evaluation at our institution. On review of his MRI, we identified the lesion as a herniation of the mesial temporal lobe on the left side, such that part of the mesial temporal lobe was in the posterior fossa, adjacent to the brainstem. Markedly absent were oculomotor nerve palsy or other hallmarks of compression.

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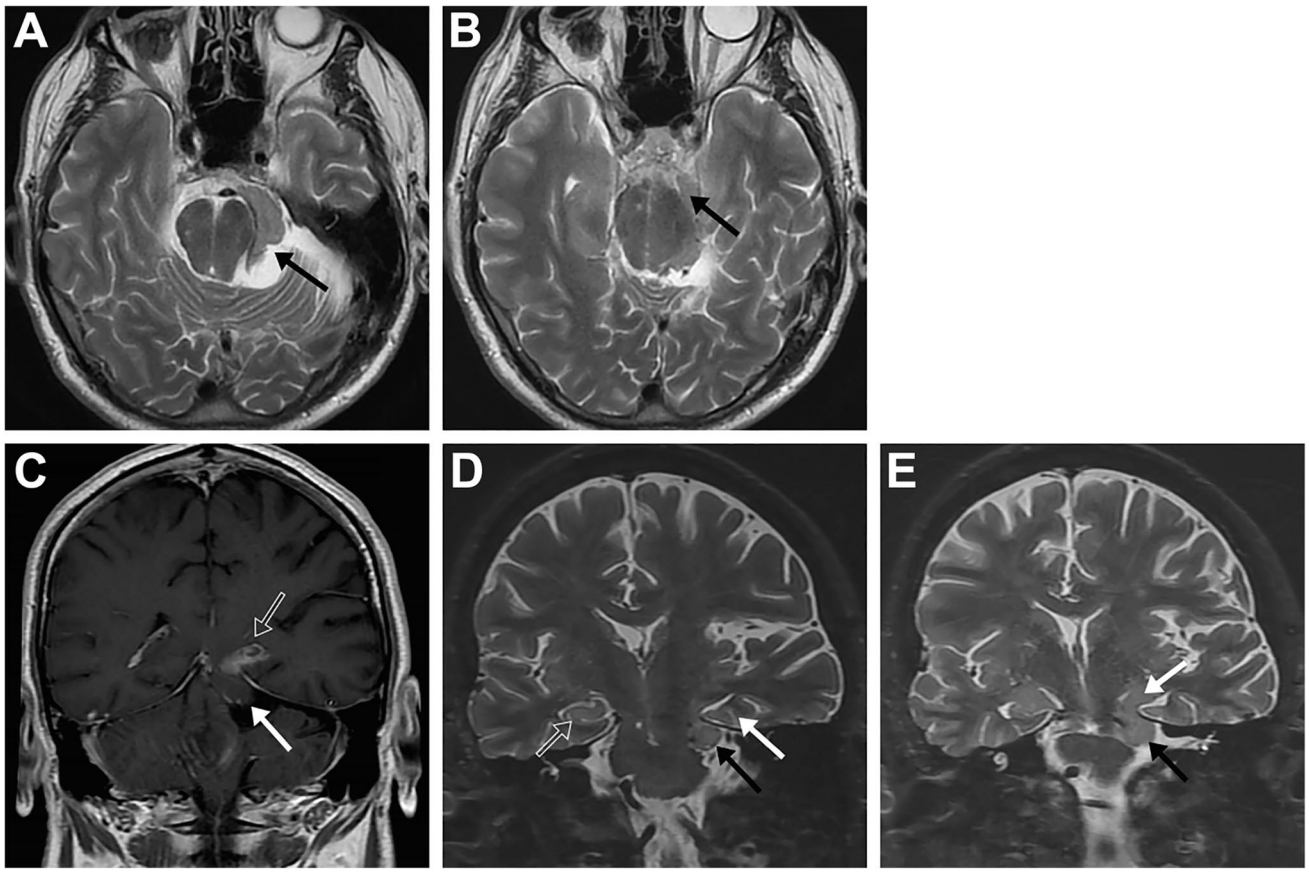

Fig. 1 **A** Axial T2-weighted MRI from inferior to superior at the level of the midbrain of the left temporal lobe herniation shows an apparent soft tissue mass along the left midbrain that appears mildly hyperintense to gray matter (black arrow). **B** Axial T2-weighted MRI from inferior to superior at the level of the junction of the thalamus and brainstem shows the "mass" (black arrow) related to herniation along the medial margin of the tentorial margin with mass effect on the adjacent brainstem. **C** Coronal T2-weighted MRI with fat saturation shows the mesial temporal lobe herniation (open arrow) with local mass effect on the adjacent brainstem. Note the continuity of the "mass" with the remainder of the left mesial temporal lobe (white arrow). **D** Coronal T2-weighted MRI with fat saturation at the level of the hippocampus shows a normal hippocampus on the right (open arrow) and lack of the normal hippocampus on the left (white arrow). Note the herniated mesial temporal lobe beneath the tentorium (black arrow). **E** Coronal T1-weighted MRI with contrast shows the herniated mesial temporal lobe (white arrow) within the posterior fossa. Note the inferior displacement and partial herniation of the left choroid plexus (black arrow), which contributed to the appearance of a "mass."

The patient had no history consistent with CSF leak or traumatic brain injury per se. He had sustained head trauma several years earlier, when a piece of metal traveling at high velocity entered his left face and exited his right eye. The eye was not salvageable, and he had a prosthetic eye implanted.

His past medical history was significant for a seizure at age 4 years. He was medication free without further known epileptic events until he had another seizure in high school. Antiepileptic medications were started at that time. Approximately 15 years before his presentation, he was noncompliant with his medications for a period, resulting in a tonic–clonic seizure with status epilepticus, and he required extended hospitalization. He has had cognitive impairment with poor short- and long-term memory recall since then and requires assistance with all activities of daily living.

Approximately 8 years ago, he experienced a several-week period of severe daily headaches without noticeable neurologic change, but he has not had episodes of severe headache since then. Over the 3 months before presentation, he experienced infrequent mild headaches. He continued to have infrequent seizures that were not tonic–clonic in nature. Other symptoms included bladder and occasional bowel incontinence for 1.5 years and ongoing dysphagia for ~ 8 years; these symptoms prompted the brain MRI.

After the lesion was properly diagnosed, the patient was further evaluated by our epileptologists and diagnosed with left mesial temporal lobe epilepsy (TLE).

The patient gave consent for publication.

Discussion

Transtentorial uncal herniation is a relatively common entity usually found after trauma or stroke. The Monro-Kellie doctrine explains why many acute changes in compartmental intracranial pressure can result in acute brain herniation from one brain compartment to another [6].

Acute uncal herniation often presents with hemiparesis, ipsilateral third cranial nerve palsy, and coma and, if left untreated, typically leads to death [4]. First-line imaging in an acute setting involves a head computed tomography scan to identify a process that may require surgical intervention. Intracranial pressure monitoring, medical management, and ultimately surgical intervention, such as decompression, are often required emergently [9].

Chronic herniation has been associated with CSF shunting and leaks, embryologic processes such as Chiari malformation, trauma, hydrocephalus, pneumocephalus, edema, or tumor. Although occult herniation has been described in children, chronic herniation of the temporal lobe in an adult without underlying pathology is rare and not well described in the literature. An asymptomatic parahippocampal temporal lobe herniation believed to be embryologically related has also been described [3]. Seizure, particularly with status epilepticus, resulting in tonsillar or uncal herniation, is usually a sequela of resulting cerebral edema and is usually fatal [4, 8, 14]. When the resultant edema is addressed or resolves, the herniation can correct itself [8, 14]. To our knowledge, there has not been a symptomatic case reported without prior known risk factors of ongoing intracranial pressure change.

TLE describes various pathologies relating to changes in sites within the temporal lobe causing seizure. It is the most common form of focal epilepsy [10]. It often presents with an aura and can significantly affect cognition and memory [1]. The seizure can progress to tonic–clonic status or remain localized and cause mild impairment and automatisms. None of the symptoms associated with TLE are pathognomonic, and diagnosis is largely clinical with supportive electroencephalogram and MRI findings, but the latter are often subtle, making definitive diagnosis difficult. Our patient's chronic seizures, with onset at age 4 years, could be secondary to his temporal lobe herniation, but, given the long history and presumed workup, this is unlikely.

The cause of our patient's left (dominant) temporal lobe herniation is unknown. The temporal lobe may have herniated during the status epilepticus event and hospitalization 15 years ago without being noted at that time. Another unlikely possibility is that this chronic herniation has been present from birth and is embryologically related (such as cortical dysplasia). This second hypothesis is much less likely because the patient had normal functioning prior to his status epilepticus event and had marked decline in function following that event. This sequence of events is better explained by an event leading to mesial temporal lobe herniation. Without access to prior imaging, we cannot definitively confirm the cause of herniation. The lesion is not a tumor; furthermore, it would be extremely rare for a small tumor to cause that degree of transtentorial herniation. Neither the patient nor his family was aware of this pathology despite prior workup with imaging.

Conclusion

Chronic temporal lobe herniation without a known underlying cause is rare and may be associated with underlying mesial TLE. This case demonstrates that this rare occurrence may be misinterpreted as neoplasm on imaging.

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Declarations

Ethical approval All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Conflict of interest The authors declare no competing interests.

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